nous epinephrine, diphenhydramine, aminophylline, and methylprednisolone sodium succinate for presumed anaphylaxis to the liniment used in the acupuncture. A chest x-ray study was done that showed bilateral, nearly complete pneumothoraces. Bilateral chest tubes were placed, which resulted in a prompt resolution of the hypotension and respiratory distress. He was discharged well several days later and was thought to have no underlying pulmonary disease.

This case, and that reported by Dr Wright and associates, indicate a potentially lethal complication of thoracic acupuncture. I am also aware of a number of patients who have had unilateral pneumothorax related to attempted corticosteroid injection of the shoulder or thoracic trigger points. Practitioners in any field of medicine who use thoracic injection techniques must exercise caution in the depth of insertion and be aware of the potential for pneumothorax.

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Carbon Monoxide Poisoning

TO THE EDITOR: We read with interest the report by Gasman and colleagues regarding an indoor barbecue and carbon monoxide poisoning in the December 1990 issue.[1] We have encountered a similar group of patients that underscores some of the authors' points:

Two 31-year-old non-English-speaking women were brought to the emergency department by paramedics, who suspected the patients had food poisoning. They had abdominal pain, nausea, vomiting, dizziness, and headache. One had had a single episode of diarrhea. They had shared a meal of barbecued beef. Three other members of the household had experienced similar but milder symptoms that did not require treatment.

The results of a physical examination of both patients were normal. On further questioning, the women admitted that the beef had been prepared on a barbecue stove in their closed apartment. They had been reluctant to provide this information to the paramedics because, having been in the United States for only a brief period of time, they assumed the paramedics—because of their uniforms—had police functions.

Once this item of history had been obtained, the patients were placed on 100% oxygen therapy. Carboxyhemoglobin levels were measured and found to be 38.3% and 31.9%. The other members of the household were called and evaluated. All had normal findings on physical examinations. Carboxyhemoglobin levels were 25.5%, 21.2%, and 18.5%.

These cases underscore the similarity that carbon monoxide poisoning may display to food poisoning, especially when more than one patient is involved. The difficulty of obtaining an accurate history because of language barriers and social background may enhance the challenge of arriving at the correct diagnosis.

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1. Gasman JD, Varon J, Gardner JP: Revenge of the barbecue grill—Carbon monoxide poisoning. West J Med 1990; 153:656-657

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TO THE EDITOR: We read with great interest the report by Gasman and co-workers regarding carbon monoxide (CO) poisoning in a family using charcoal for indoor cooking purposes.[1] The article did an excellent job of highlighting a recurrent and insidious environmental health hazard. The authors made a few technical points, however, that merit further comment.

The authors quote a normal range for carboxyhemoglobin (COHb) saturation of less than 0.150 (<15%). Most observers agree that COHb levels as low as 10% to 15% can be responsible for headaches and dizziness. A 1989 study implicated carboxyhemoglobin levels as low as 2% in reducing exercise tolerance among patients with coronary artery disease and angina.[2] Further, in a large population-based survey, 95% of nonsmokers were found to have COHb saturations below 0.02 (<2.0%) and 95% of smokers below 0.085 (<8.5%).[3] Hence, more reasonable "normal" ranges are 0.02 or less (≤2.0%) for nonsmokers and below 0.09 (<9.0%) for smokers, with an irreducible minimum of 0.003 to 0.005 (0.3% to 0.5%) due to porphyrin catabolism.

The authors also state that dissolved CO combining with cytochromes, not impaired oxygen delivery due to COHb formation, is responsible for the toxic effects of CO. They base this assertion on a 1976 report in which anemic dogs transfused with CO-saturated blood failed to show signs of CO toxicity. In studies of experimental animals perfused with a hemoglobin substitute (a fluorinated compound without special affinity for CO), however, the animals tolerated atmospheres of 3% to 5% CO—environments that would have been rapidly fatal had the primary mechanism of CO toxicity been the interaction of dissolved CO with cytochromes.[4]

The authors make the point that "patients with severe neurologic or cardiovascular symptoms or very high COHb concentrations would benefit from hyperbaric oxygen." Although hyperbaric oxygen does substantially hasten the elimination of CO and the reduction of COHb levels in CO poisoning, there have yet to be any controlled studies showing that the ultimate outcome in patients treated with hyperbaric oxygen is better than in patients treated with normobaric oxygen.

Concern over the type of event outlined in Dr Gasman's report prompted the California Department of Health Services to issue a public warning in January 1990 cautioning against the use of gas ranges and unvented gas or kerosene heaters for indoor heating, as well as the practice of using charcoal for indoor cooking. The advisory especially targeted Asian immigrants because the traditional use of charcoal for indoor cooking purposes has been previously reported within this community. We are appreciative of Dr Gasman and colleagues for again bringing this issue to the forefront.

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Drs Gasman and Varon Respond

To the Editor: We thank Drs Shusterman, Liu, and Kizer for their useful comments. We agree that recent data suggest that COHb levels as low as 0.10 (10%) can be symptomatic and may account for a considerable number of ambulatory presentations. Regarding the mechanism by which carbon monoxide exerts its toxic effects, certainly the primary role is through impaired oxygen delivery due to the higher affinity of carbon monoxide for hemoglobin. We agree that no data suggest that the ultimate outcome would be improved through the use of hyperbaric oxygen; after all, most patients recover completely. Seriously ill patients would recover more rapidly if hyperbaric oxygen were available and employed, however.

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Reserpine as Monotherapy for Mild Hypertension?

To the Editor: Dr Pérez-Stable has done an excellent job of reviewing the management of mild hypertension in his article in the January 1991 issue.[1] There was no mention, however, of reserpine, which remains a very useful and cost-effective antihypertensive, albeit one no longer in style. It has been well established that reserpine is highly effective for mild to moderate hypertension and that its side effects compare favorably with other antihypertensives.[2] The oft-repeated warning that reserpine be avoided because of the excessive danger of depression has been put to rest by several studies.[2,3] In fact, reserpine has even less adverse effects and remains effective when used in doses of 0.125 mg daily as shown in the VA Cooperative Study.[4]

Although Dr Pérez-Stable states that "the cost of daily drug therapy need not be a predominant determinant of choosing a regimen," this may not be convincing to the patient who has a choice between reserpine, which costs less than $15 per year (total annual cost of both reserpine and thiazide should be less than $50), and angiotensin-converting enzyme inhibitors and calcium entry blockers, which may cost as much as $700 per year. Dr Pérez-Stable does mention that compliance may be related in part to the cost of medication but then unfortunately omits the least expensive antihypertensive available. Of course, it is of great benefit to clinicians to have a large number of new antihypertensives available, but that should not preclude the consideration of using an older, less fashionable drug, particularly when it has been shown to be highly effective in carefully designed prospective double-blind studies.[2] If, in addition, the medication has the special advantage of being very inexpensive, requires only one pill per day, and has a very low incidence of side effects,[5,6] I should think it (reserpine) deserves to be mentioned, even if briefly, in an otherwise excellent review article.

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Dr Pérez-Stable Responds

To the Editor: I appreciate the comments made by Dr Feigenbaum, which are worth noting. Reserpine is an inexpensive medication with limited adverse affects when used in low doses. I concur that the risk of depression has been overstated. Reserpine, however, is not included in the Joint National Committee on Detection, Evaluation and Treatment of Hypertension's list of first-line pharmacologic treatment. Used as monotherapy, reserpine is not as effective as the four types of medications that I discussed, and, therefore, I excluded it from the review. Similarly, I did not discuss other groups of antihypertensives such as α_1 blockers or central adrenergic inhibitors that may occasionally be effective as monotherapy. In practice, I have found reserpine to cause fatigue and nasal congestion, which limited its usefulness. Although in combination with a thiazide diuretic, reserpine is useful, I doubt that it will ever regain a premier position in the antihypertensive armamentarium.

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